

EPIDEMIC INFLUENZA.

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## EPIDEMIC INFLUENZA.

The subject of Epidemic Influenza is of interest to me for several reasons. Firstly because I had never seen a case before the 1918 Epidemic, and as a student I do not believe we were taught much about it. Thirdly because of all deaths that I have had to certify Epidemic Influenza easily holds the first place as the primary cause of death. That emphasis as to its gravity is necessary is proved by its frequent recurrence only to be forgotten as soon as it has subsided.

This is all the more noteworthy when one reviews its history for which the following authorities have been consulted:-

Handbook of Geographical and Historical Pathology:

A. Hirsch.

History of Epidemics in Britain: C. Creighton.

System of Medicine. Vol I. Allbutt & Rolleston,

"Influenza" by Sir James Goodhart.

Encyclopaedia Medica: "Influenza" by Sir J.W. Moore.

Nothnagel's Practice: O. Lichtenstern, "Influenza".

Creighton points out that in early records there was great difficulty in differentiating between ague and/

and catarrh, and that the epidemic mentioned by Hippocrates and Livy occurring in 412 B.C. was influenza.

There were epidemics from the 6th to the 10th Century A.D. but these were not considered characteristic enough by Hirsch who takes as the first authentic one which began in December 1173. This occurred in Italy, Germany and England and caused many deaths.

In October 1427 there occurred an epidemic (fatal to many) which is mentioned in a record kept at St Albans (1423-1431). This is considered by Creighton to be influenza. It also occurred in Paris the same year.

Hirsch tabulates 299 epidemics as happening between 1173 and 1875, but these include small local epidemics as well as pandemics. Lichtenstern gives the following list:-

1510. Widespread epidemic over Europe.

1557. Epidemic spreading from Asia through Europe.

Creighton quotes a writer, Wriothsesley, who states that in 1557, "This Summer reigned in England divers strange and new sicknesses taking men and women in their heads; as strange agues and fevers, whereof many died".

This epidemic continued into 1558.

1580./



1580. The first real pandemic spreading over the East, and then through Europe and North Africa.

There are only scanty references to epidemics during the seventeenth century. A great epidemic is reported over North and South America during 1627.

Creighton mentions an account by Willis of an epidemic in England during 1657 recurring during the Spring 1658 and Autumn 1658, the middle one he considers to be influenza, and mentions that this account is the first piece of epidemiology written in England.

Evelyn in his diary states on October 15th, 1675, that there was an epidemic "rife all over Europe like a plague."

1709-12. Widely distributed epidemics with a low mortality.

1729-33. Spread from East to West in two waves - 1729 and again 1732. Hirsch states that there was a diffusion all over the world.

1743. Spread from the Baltic west and south.

1757-8, 1761-2 and 1767. Epidemics in North America with spread to Europe.

Heberden read a paper in August, 1767, at the Royal College of Physicians on "The Epidemical Cold in June and July, 1767."

1781-2./

- 1781-2. A great pandemic beginning in China and India and spreading through Russia all over Europe and Africa. The College of Physicians formally adopted the name Influenza though the name had already been used in 1729 in England in an account of an Italian Epidemic.
- 1788-90. Another pandemic period again beginning in Russia and spreading over Europe and America.
- 1799-1803. Several outbreaks beginning in Russia in 1799 and becoming pandemic.
1827. Diffused through Siberia and Eastern Russia.
- 1830-3. Severe pandemics, first beginning in China and reaching Europe through Russia 1831, second 1832 a very intense outbreak also travelled from East to West, the third 1833 spread the same as the first.
- 1836-1837. Beginning in Australia, Java and India, it spread to South Africa; from the East to Russia thence west all over Europe.
- 1847-8. A pandemic over Europe and Russia and reaching North America in 1848.
- 1889-90. A very extensive and severe pandemic beginning in Turkestan reaching Europe through Russia, it spread absolutely all over the world.

There were small recurrences of the disease for the next fifteen years then all was quiet till 1918 when the worst one of all swept over the world.

Sir John Moore says of this epidemic:-

"It has fallen to my lot to witness a Cholera Epidemic in 1866, smallpox epidemics in 1872 and 1878-1880, and last, not least, the epidemic (or, rather pandemic) of influenza in 1889-90. But, in my opinion, none of these visitations equalled the present outbreak in extent, virulence, or treacherous course."

In South Africa, I may add, we had a terrible visitation, but such is human nature that it is now almost forgotten, even amongst the medical profession.

There are some who say that nothing further was learnt about the disease, but I am of opinion that some advance has been made, if not about its etiology, at least about the prophylaxis and treatment of its complications.

The disease itself, may be described as an acute and very infectious fever, with a very short incubation period (12 to 48 hours). It has a very sudden onset, most often without any preliminary feeling of "out of sorts". The patient has a feeling of chilliness, often a sense of weakness in the legs, giddiness, sometimes they collapse. Many have the disease without feeling ill/



ill at all. In nearly all cases there is a rise in temperature, and the pulse rate does not rise with the temperature, which lasts for 3 or 5 days. Often there is a great deal of sweating, the perspiration and breath having a peculiar foetid odour (I know of no other similar odour). The tongue is coated and often remains coated for many months afterwards. There is a paralysis of the vasomotor system and a great lowering of the patient's resistance which leads to the many complications which are the ultimate causes of the high death rate.

The disease itself confers a lasting immunity, though the lowered resistance to other infections may persist for a long time.

In South Africa young adults especially were attacked in the 1918-1919 epidemic, old people almost entirely escaping. Children became infected mostly after the epidemic and then suffered mostly from the gastro-intestinal form, though this should really be regarded as a complication. I attribute the immunity of the older people to the 1890 epidemic and this brings us to the question as to its etiology.

Dr W.D'Este Emery (Practitioner, February 1919) mentions that at one time (1892) the cause was thought to be Pfeiffer's bacillus but that observations made in/

in England, France, Germany and America have shown that in attacks clinically typical Epidemic Influenza the bacillus was not found. This was first pointed out in England by Bulloch (B.M.J. April 26th 1905). Dunn and Gordon (B.M.J. Aug. 26, 1906) published an account of an epidemic like influenza with an absence of Pfeiffer's bacillus and frequent presence of *Micrococcus Catarrhalis*. Besançon and de Jong (Bull. soc. med. hôp. de Paris, March 2-16, 1905) in investigating an epidemic found Pfeiffer's Bacillus present in only few cases, pneumococcus almost constantly and occasionally other organisms such as *Pneumobacillus Streptococci*, pseudo-Diphtheria bacilli etc., in the majority of cases they also found *Micrococcus Catarrhalis* or an organism resembling *M. tetragenesis*.

Emery mentions an epidemic which occurred in America in 1915-16. According to Mathers, it "resembled in every detail epidemic influenza" and was different from common acute rhinitis; there was a high death rate. The point about this epidemic is that the predominating organism was a *Streptococcus*, an organism which had often been recorded as occurring in influenza, but never so commonly, and this is of importance in that, in the severe cases which were seen generally in the last epidemic, the organism is the most important because the most fatal of the associated organisms. It does not occur in all cases, even/



even in fatal ones: we still see pneumonia and bronchopneumonia proving fatal when only Pfeiffer's bacillus, the pneumococcus, and *M. catarrhalis* are present.

In most cases, however, which tend to be fatal and more especially in the fulminating or septic cases streptococci form the predominating infection, and may be pure, or apparently so, both in the lungs and in the blood.

In addition to these streptococci which, from their pathogenic powers, constitute so important a feature in this present epidemic, it seems to me certain that pneumococci are more prevalent than in former epidemics. Mathers found them in 49.1% of his cases, but not usually pure, and he adds that the biological forms, which were more common, in ordinary lobar pneumonia, were not those which predominated in the epidemic. "Emery points out that the organs of patients who died in 1890-2 from the epidemic of that period, preserved in museums, show an essential similarity with those who died in 1918 epidemic and that the clinical differences which were seen in some cases are not more than one might expect from differences in the secondary infections. He further states that the influenza bacillus does not cause a short, sharp infection with complete and permanent immunity. There is experimental evidence to show that it is extremely difficult to immunize animals to its action, and in man it often causes chronic infections, which may/

may last for months or years, or which may often relapse; the same is true of the pneumococcus, streptococcus and the other organisms found in the disease. Looking at the matter from the view of the long-lasting immunity, everything seems to point to the fact that we have to deal with one of the diseases of unknown origin similar to scarlet fever, small pox, measles etc."

We must therefore assume that just as the unknown virus of scarlet fever renders virulent the streptococcus of the mouth, so the unknown virus of influenza can render virulent streptococci, pneumococci, or other pathogenic organism which may be present.

As it is due to these other organisms that complications arise and cause the high death rate, all that can at present be done is to combat these.

#### COMPLICATIONS.

Presuming that the cause is an ultra-microscopic microbe and the path of infection is through the nose and mouth, it is here also that the other organisms mentioned are harboured. The respiratory system was the most often affected.

A congestion of the nose or rhinitis was not frequent in South Africa but one might expect this to have been fairly frequent in England with its/

its wet climate.

Epistaxis, however, was very common; at least every fifth case I saw had bleeding from the nose. In not a single case did this lead to a fatal termination, and further it was noticed that of patients suffering from other severe complications nearly everyone recovered if there was at least a moderately severe nose bleeding. Discharge from the nose was very seldom present, and never copious such as happens in epidemics of ordinary colds. Sinus infections as a sequel occurred fairly frequently. I had six cases which necessitated operative interference, and that it was very prevalent one can infer from the frequency with which the disease was attended by headaches.

Similarly otitis media occurred more frequently than was suspected. I came across four cases where the condition had extended to mastoid suppuration needing operation.

Tonsillitis was very common, so also quinsies. Immediately after the epidemic I had very often as many as three tonsillar abscesses to open in one day. In one case during the epidemic where this had been the only complication, and where the patient was already convalescent, I saw the patient die suddenly.

The trachea and larynx were probably more often involved, together with a more general inflammation, but that these parts sometimes were principally involved /



involved can be inferred from a troublesome cough and hoarseness where no other signs and symptoms were found; there is the possibility that pus from the accessory sinuses may have been the irritating factor.

The bronchi and lungs were most often involved and this was the most fatal complication, death taking place very rapidly.

There was often capillary bronchitis, broncho-pneumonia and a few cases of lobar pneumonia.

If a patient was not over the influenza attack after five days, the temperature remaining up and the pulse becoming rapid, there was nearly always some complication with involvement of the lungs. Very often during the first two or three days this complication had already begun, and ended in death.

The physical signs varied so much that one could not rely on them to make a diagnosis of pneumonia. Bronchial breathing was often absent, probably due to a filling up of bronchi supplying the affected lung tissue. On percussion dulness was often slight and patchy. One heard moist sticky râles in nearly all cases. There was a great tendency for the inflammation to spread from one lung to another, and from one lobe to another, the one lung clearing up whilst the other took on the inflammation. With us the apices were most often involved. My colleagues in our town told me that such was also their experience. The points/

points one had to rely on generally were: the temperature remaining high and irregular, the breathing becoming more rapid, more marked cyanosis, patient becoming restless, may be delirious (it is remarkable, however, how many patients retained their full mental faculties right up to the very moment when they died) in fact the general condition of the patient becoming worse was enough to suspect a complication especially of the lungs. Regarding cyanosis, I saw very few patients with marked cyanosis recover, death generally taking place from a profound toxemia; one could smell them on entering the room. Where recovery took place this was generally by lysis.

Cough was often slight, sometimes absent; sputum was sticky sometimes mucopurulent. In many cases there was no expectoration.

Regarding the pathology of these cases I did not see any post-mortems, the morbid changes described by Hector McKenzie (Practitioner, Feb. 1919) are such as one might expect to find in the broncho pneumonias. He says:- "The most common morbid change in the lungs is broncho-pneumonia. It was present in 54 out of 76 fatal cases examined during the recent epidemic in the British Armies in France. Numerous scattered areas of lobular consolidation may be seen, affecting an extensive tract but separated by small air-containing patches. /

patches. The cut surface is somewhat mottled and smooth or slightly granular. The consolidation is due rather to a cellular inflammation than to a fibrinous exudation. The solid areas more resemble spleen than liver substance, and the lung may be said to be splenecized instead of hepatized. Pfeiffer described small patches of consolidation, from the size of a pin's head to that of a pea, separated by areas of a greyish-yellow colour from the surrounding dark red tissue. At the centres of the consolidated areas, yellowish-grey, thick and very tenacious pus may be seen exuding from the cut sections of the bronchi.

Not only Pfeiffer's bacilli, but pneumococci, streptococci, or staphylococci may be present in the consolidated islets, some within, some outside the leucocytes and the epithelial cells. In some cases, and this has again been observed in the epidemic in France, the pneumonic areas are small and shotty, closely resembling miliary tubercles. The microscope showed these to consist of consolidated lung with fibrinous exudate surrounding inflamed bronchioles. This form has been appropriately called 'miliary pneumonia'. At a later stage small abscesses may form. Some lobular areas are entirely filled with leucocytes and, merging into one another, form small centres of suppuration.

Subpleural/



Subpleural and interstitial haemorrhages are observed in some cases. These, however, are usually small and localised.

Pneumonic consolidation of the lobar type is also met with, but is much less common than the bronchopneumonic form. The appearances of the consolidated lung resemble those of ordinary pneumonia. The upper lobes seem to be more often affected than in primary pneumonia. The pneumococcus or the streptococcus pyogenes may be found to be present.

Influenzal pneumonia is sometimes slow to undergo resolution. The affected areas may be transformed into cicatricial connective tissue, and be replaced by firm greyish-red fibrous masses of almost cartilaginous consistence. Gangrene of the lung sometimes occurs.

The pleurae are frequently affected. When there is effusion (seldom a large one) it is more commonly seropurulent than purulent. It has a peculiar, cloudy, rather yellow appearance and has been likened to clay water, or a suspension of cream of tartar."

This slow resolution was at first a matter of difficulty. In four cases of a colleague of mine he had asked me to see, thinking he had to deal with an empyaema, aspiration was negative. I had also found this in several cases. All these cases made a good recovery, some however having an irregular temperature, and/

and dulness for three weeks to a month. One patient of mine could not be convinced that he did not have an empyaema and persuaded me to resect, this was done under local anaesthesia. There was no fluid nor pus, but the lung was of a firm cicatrised consistency.

Chronic affections of the lungs did not occur; this I attribute to our climate. The prognosis in the lung complicated cases was always grave, especially in the country patients; so many people were affected that voluntary nursing was very scarce, and housing conditions were poor. Medical visits were often limited to one or two occasions; 40% of these cases died.

The outlook in the town cases was very much better, as nearly every serious European case was treated in a large airy school which had been converted into a temporary hospital. Four churches served a similar purpose for the town native population. No more than 15% of these ended fatally.

As to treatment, at first vaccines were not included, and treatment consisted of plenty of fresh air, sponging with cold water, a nose and mouth wash with a weak chinol solution, applications of antiphlogistine, and Nux Vomica in the hope of keeping the patient toned up, (this was found more useful in after treatment), Digitalis in the form of Digalen, but/

but as it did not seem to do any good was discontinued, a cough mixture of Ammonium Carbonate and Vinum Ipecacuanha, with the addition, in some cases of sticky expectoration, of Potassium Iodide; no alcohol, no opium, Isobromyl as a sedative.

Diet consisted of milk and eggs, finely chopped meat, rusks, and as much water as possible. Where the patient could take a large amount of water a pinch of salt and a good deal of sugar was added. Hypodermic Injections of Camphor dissolved in ether and oil, also injections of Pituitrin, Pituitrin and Adrenalin. These latter, I am of opinion, tided over some very bad cases.

Later stock mixed vaccines were used with much better results.

Later still I had the opportunity to use Diénol (a French preparation of Colloidal Manganese). The use of this was very marked and gave one much greater confidence in meeting an acute lung condition. I have since used it in all cases of Bronchopneumonia and Pneumonia with very marked success.

With regard to early vaccine treatment, I am thoroughly convinced of its efficacy in practice and hereby give some figures, the results of Dr W.H. Wynn (Practitioner, June 1919).

"107 consecutive hospital patients injected during the first five days of their illness. 28 were injected on the first day, 23 for the first time on the/



the second day, 22 on the third day, 20 on the fourth and 14 on the fifth. Cases of simple uncomplicated influenza are excluded. Several were admitted in an apparently hopeless condition, many suffered from the most severe type of the disease with cyanosis.

The term 'bronchopneumonia' is here held to include all cases of pulmonary complications for want of a more inclusive word. Every type of lesion was found - Bronchiolitis, extreme congestion, collapse, oedema, pleurisy, consolidation. Several had extensive confluent broncho-pneumonia, and others would be more correctly classified as influenzo-pneumococcal or streptococcal septicaemia. The extent of the lesions naturally varied with the day of admission, the early cases showing, may be, only some diminished resonance at the bases of the lungs, with faint breath sounds and numerous crepitations or rhonchi. With each day's delay the cases became more severe, but these later cases had passed through the earlier stages, and the object of treatment during the first day or two was to prevent the onset of graver symptoms. In the majority there was no preliminary period of simple influenza, pulmonary signs were present from the beginning. In a few, there had been a simple influenza followed by a period of normal, or nearly normal, temperature before the sudden onset of complications. In these cases, the day of the disease/

disease has been reckoned from the beginning of the secondary fever.

The vaccine used has been a polyvalent triple vaccine containing equal numbers of pneumococci, streptococci and influenza bacilli. Some of the earlier cases were given a smaller dose of influenza bacilli than the other two, but there was no good reason for this. All the pneumococci were obtained from primary cultures and the streptococci mainly from primary cultures. It is important that the dose of vaccine should be adequate; for an adult man, 80 to 100 millions of the three organisms; for an adult woman, 60 to 80 millions; for a child of 12 to 14, 40 to 50 millions; for a child of 2 to 3 years, 10 to 20 millions.

Patients inoculated during the first day - 28 - all recovered; in 20 the temperature fell to normal in 24 hours, in a few within 12 hours; in 4 the temperature fell in from 24 to 48 hours. In one there was a fall to normal in 24 hours but not complete defervescence till the 4th day. In 2 there was complete defervescence on the 5th day. In one the fever lasted 7 days. This was a woman with severe confluent broncho-pneumonia. The first dose was 30 million Influenza bacilli, and 50 million each of streptococci and pneumococci. After a dose of 60 million influenza bacilli and 80 million each of the others the temperature/

temperature fell from  $103^{\circ}$  to  $101^{\circ}$ . It rose again to  $104$  and on the fifth day a dose of 80 million of each organism was given and caused a more effective response.

Patients injected on the second day - 23 - ; in 11 the temperature fell to normal in 24 hours, 2 normal in 48 hours, 6 within 3 days, 2 within 4 days and in 2 the fever persisted more than 4 days, the preliminary fall being followed by rises which necessitated further injections.

There was one death in this group, a woman with violent mania, extensive broncho-pneumonia, who after a dose of 50 million each became rational in 36 hours and whose temperature dropped to normal in the same time and remained so till the 8th day when it rose to  $102$ . She was given a second injection of 50 millions followed by a fall to  $99$ , but on the following day she died suddenly of pulmonary embolism.

Patients injected on the third day - 22 - ; in 11 (50%) temperature fell to normal in 24 hours and in 72.7% the temperature reached normal in 48 hours. In 20 there was complete defervescence in 4 days. Two patients died.

Patients injected on the 4th day - 20 cases ; in only six did the temperature become normal in 24 hours, 8 in 48 hours, and in 10 by the end of the third day. Half of this group required more than one/



one injection. All the patients in this group were of the worst type. There were 5 deaths.

Patients injected on the fifth day - 14 cases. In 5 the temperature reached normal in 24 hours, in 4 more in 48 hours. This group was one of great severity. There were 2 deaths.

An analysis of these cases emphasizes the importance of early treatment. Whilst in 71.4 per cent injected on the first day had a normal temperature within 24 hours, on the second day the percentage had fallen to 47.8, on the third day 50, the fourth day 30, and the fifth day 35.7. Of the total 107 cases, however, the temperature fell to normal within 24 hours in 50 per cent. In 65 per cent of the total number the temperature became normal within 48 hours. The 10 deaths give a rate of 9.1 per cent. Seven of the 10 were not inoculated till the 4th or 5th day. The only death during the first 2 days was from pulmonary embolism after apparent recovery from the pneumonia and acute mania. Of the others, four were complicated respectively by mitral stenosis, advanced pregnancy, meningitis and asthma."

Regarding the cardio-vascular system one expects to find that like all fevers the heart must be affected, temporarily, at least; thus one finds some increase in size, the heart sounds become soft and/

and there may be systolic murmurs. Unlike other fevers one mostly found the pulse slow in spite of the rise in temperature and I accounted for this that it was due to the toxin acting on the nervous system, producing a stimulation of the vagus. I had many cases of an ordinary severity with no lung complication where the patient complained of feeling his heart stop, especially when about to fall asleep. This passed off in most cases in a matter of 3 weeks.

I am certain that the myocardium, including the auricular-ventricular bundle, suffered permanent damage in some cases of simple influenza, undoubtedly in cases complicated by other organisms, such as in quinsies and broncho-pneumonias. I can remember six cases of sudden death after the epidemic, three of these had no complications, two had tonsillitis with abscess formation, and one had broncho-pneumonia. I mention these cases because I know that the heart condition was sound previous to influenzal attacks. Three of these patients complained every now and then of a feeling as if the heart had stopped. I attended inquests on three other sudden deaths, where I was satisfied that the cause of death was primarily influenza with a secondary involvement of the heart muscle.

In no case during the epidemic did I find influenza causing a valvular lesion.

Of/

Of the six cases mentioned, one is permanently impressed on my memory, for I had passed him first class for insurance in 1921 and he died suddenly in 1925 after lifting a heavy weight.

Many patients after having had influenza complained of weakness and shortness of breath on exertion, but one could detect nothing wrong with the condition of the heart. A tonic including nux vomica, fresh air and moderate exercise short of causing exhaustion always produced the necessary improvement and recovery to apparently good health. But looking back makes one more cautious as to a good prognosis and I am sure many of the sudden deaths years after can be really put down to an already long forgotten attack of influenza.

As to the nervous system, this is necessarily involved to a moderate and temporary extent in practically every case. The feeling of weakness, sleeplessness, sweating, loss of taste and appetite, headache (this latter may be ascribed to sinus infection) are all due to involvement of this system.

The rarity of the graver complications, at least in our district, was remarkable, where among the native population syphilis is very prevalent. (In passing I may mention that in the ordinary course of practice I came across only one case of syphilis of/



of the central nervous system with symptoms, and this is the more remarkable for the reason that the natives do not as a rule submit to thorough and early treatment. This fact was also noted by a colleague who has been in practice there for over thirty years.)

There were four cases of insanity, none of which had had syphilis. One case ended in suicide and the other three recovered; one of these had a family history (brother) of insanity.

I had one case of paralysis of the soft palate with recovery after three weeks.

Two cases of paralysis with apparent unconsciousness. I could not make out anything definite at examination because the patients (adults over 50) were both unconscious. I could get no response to the reflexes, no response to painful stimulation, but as both made a rapid recovery I can only believe the condition must have been hysteria.

During 1925 we had an outbreak of gastro-intestinal influenza and the patients chiefly affected were children, of whom many died. Adults however were also affected. The onset was sudden generally, with some slight fever, then vomiting and severe watery diarrhoea followed later by some enlargement of the liver and slight jaundice. The disease was prevalent throughout the country. Durban, Johannesburg and Bloemfontein were all affected at the same time.

During/

During this small epidemic I had five cases in adults where paralysis followed an acute gastrointestinal disturbance. Three of these made a complete recovery, one partial, and the other a male of 66 years, died. It was my opinion that the lesion was in the form of haemorrhage into the cord in 3 cases, (paraplegias without pain); in the other two into the cortex, one was a monoplegia and the other a hemiplegia. I saw only one case (1919) where the haemorrhage was outside the membranes; there was pressure on the nerve roots and the patient suffered intense pain. In this case there were haemorrhages into the skin early in the course of the attack, and I gave a bad prognosis; the patient died.

The outlook as to the mental cases is good, and one can expect nearly all such cases to recover; there is always, however, a danger of suicide. In cases of paralysis the outlook is also fair though an attack of influenza is apt to find out any weak spot in the nervous system.

The kidneys must also suffer in an attack in getting rid of inflammatory products and organisms, but apart from 4 cases of marked haematuria I saw no other cases where this system was principally picked out; all 4 made good recoveries.

There was one case (girl of 17 years) where the main feature was the suppression of the menses for three months. The temperature had remained irregular for/

for 6 weeks and I could find no focus to account for this.

The pregnant uterus added greatly to the gravity of an attack, 50 per cent of my cases died.

Involvement of joints with suppuration for 9 months followed one case of tonsillitis. The organisms found were staphylococci and streptococci.

In many cases, as in other fevers, there followed a temporary loss of hair; another way in which the skin was the sufferer was in the form of very obstinate sores, especially on the face. These two skin affections occurred always during convalescence. During the actual attack, however, one frequently saw a fine rash resembling scarlet fever, the rash lasting about 24 to 36 hours.

Glands like the thyroid and suprarenal were undoubtedly affected, and one can believe that like in hypothyroidism the liability to other infections were thus increased. Many people after an attack became stout, and the form the obesity took is similar to that of myxoedema in the reaction to temperature. They were comfortable in hot weather and uncomfortable in cold. This sensitiveness to cold was very marked in many who were not stout.

It will thus be seen that though the respiratory system seems to be the most likely seat of fatal complications, no part of the body escapes the disease or its effects.

Further/



Further, when one comes to consider the fact that the whole world is liable to infection at some time or other, and that this occurred fairly frequently, and will occur again, that the disease gives rise to a very high death rate, it seems strange that epidemic influenza seems to take a back seat in importance as soon as an epidemic has subsided.

Money and time is constantly being spent on research in diseases like Tuberculosis and Cancer, but to my mind something more should be done to find the cause of epidemic influenza, especially as it seems likely that with such a discovery the etiology of diseases like measles, scarlet fever, encephalitis and others will be revealed.

That some progress at least in the prevention and treatment of its fatal complications has been made, I am fully convinced, and I will strongly advocate the early use of a triple vaccine of "Influenza" bacillus, Pneumococcus, Streptococcus plus the Micrococcus Catarrhalis.

There is a common tendency, in treating a case, to exaggerate the efficacy of a particular remedy when one has had one brilliant result. There is also the tendency to condemn a remedy when it has failed after a short and unfair trial, forgetting that such a trial has often been postponed until too late.

My/

My judgment is expressed only after the experience of many cases where no vaccines were used, followed immediately by a large number of cases of a like severity treated under similar conditions plus the early use of vaccines. The results were so striking that then, and then only, did I feel that I was of any use to the Community.

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